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IN THE UNITED STATES DISTRICT COURT

FOR THE DISTRICT OF ALASKA AT ANCHORAGE

ENOCH ADAMS, JR., LEROY ADAMS, ANDREW KOENIG, JERRY NORTON, DAVID SWAN AND JOSEPH SWAN, Plaintiff, v. TECK COMINCO ALASKA INCORPORATED Defendants.	Case No. A04-49 CV (JWS) EXPERT REPORT OF JOYCE S. TSUJI

I. Introduction and Summary of Opinions

This report presents the opinions that I, Joyce Tsuji, anticipate providing in this matter. I have been requested to provide expert opinions on behalf of Teck Cominco Alaska Incorporated (Teck Cominco).

1. I have been asked to render an opinion on whether the discharge monitoring permit violations for total dissolved solids (TDS), cyanide, and cadmium from the Red Dog

Mine between August 1998 and May 2003, noted by Adams et al. in their complaint against Teck Cominco, would result in human health effects or a reasonable fear of possible future disease based on the preponderance of scientific evidence. Based on the information provided to me, evidence is lacking that documents actual human health effects caused by exposure to the constituents named in the alleged permit violations. Therefore, the primary focus of human health concerns for this legal action appears to be the fear of possible future disease associated with either the river water or fish or other animals that might accumulate these constituents.

2. Exposures of the local population associated with the alleged permit violations of TDS, cyanide, or cadmium would not result in harmful effects or elevated risk of future disease for local populations. For the named constituents, thresholds for exposure exist below which no effects are expected, now or in the future. None would be considered carcinogenic by the exposures associated with the alleged permit violations; therefore, assessing the risk of cancer is irrelevant for this case.

3. This opinion is supported by my evaluation of the available site data for Wulik River water, Kivalina drinking water, fish tissue data, and caribou tissue data, as well as by the scientific literature and regulatory guidelines and standards.

4. I reserve the right to modify and supplement my opinions as further information becomes available, including through the deposition of plaintiffs' experts, and to express new opinions in response to new information or to opinions expressed by plaintiffs' experts.

II. Qualifications and Publications

5. I am a Principal Scientist in the Health Risk division of Exponent, a multidisciplinary science and engineering consulting firm. I have a Ph.D. focused in Physiology and Ecology from the Department of Zoology, University of Washington, and have been certified by the American Board of Toxicology since 1992. I have served on various expert scientific committees for the National Academy of Sciences/National Research Council, the U.S. Environmental Protection Agency (EPA), the U.S. Army, and the State of Washington. I am a member of the Society of Toxicology, the Society for Environmental Toxicology and Chemistry, the Society for Risk Analysis, and the Society for Environmental Geochemistry and Health.

6. From 1987 until the present, I have worked as an environmental consultant, conducting, overseeing, and reviewing human health risk assessments. Particular areas of experience include exposure assessment, biomonitoring, and the toxicology and bioavailability of metals. I have designed studies of direct and indirect (i.e., food chain) exposure to metals in populations, including subsistence populations, and have developed programs for health education and biomonitoring of communities potentially exposed to metals in the environment. My experience over the past 17 years has included numerous mining and smelting-related sites involving health risk and exposure studies in the United States and in other countries. I have also served as a toxicology expert for the EPA, U.S. Department of Justice, the State of New Jersey, the U.S. District Court for the Northern District of Texas in Dallas, and for the Victoria Environmental Protection Authority in Australia on sites with potential exposure to metals. At the 2001 meeting of the

International Congress of Toxicology, I was an invited speaker in a workshop on *Risk Assessment and Risk Management for Mining in Developing Countries*. My presentation evaluated background dietary metals intake for subsistence populations in a mining area of Southeast Asia. In September 2003, I was invited to give a plenary presentation on lead exposure pathways at the lead exposure conference in Port Pirie, Australia, that was jointly sponsored by government and industry.

7. I have considerable familiarity with metals and mining sites in the western United States, including Alaska. Currently, I am serving as a senior advisor on the human health risk assessment work conducted by Exponent for the Red Dog Mine site. My qualifications and publications are set forth in greater detail in my curriculum vitae, attached hereto (Attachment A).

III. Case Information Reviewed

8. In arriving at the opinions set forth in this declaration, I have reviewed the following site and case-related information:

- Revised Complaint for Injunctive and Declaratory Relief and Civil Penalties, Case No. A04-49, U.S. District Court for the District of Alaska at Anchorage.
- Teck Cominco Alaska Incorporated Red Dog Mine NPDES Permit AK-003865-2 Reauthorization Request, February 2003.

- EPA, Environmental Assessment Red Dog Mine Project NPDES Permit Modification, Northwest Alaska, Teck Cominco Alaska, Inc. NPDES Permit No. AK-003865-3, January 2003.

- Water quality data for the Wulik River from 1998 through September 2004.

- Village of Kivalina drinking water and Wulik River Station 1 sample results from December 2002, with cover letter from R.G. Scott, Teck Cominco, to Craig Paulsen, EPA, Region 10.

- Exponent data validation report for the Kivalina drinking water December 2002 sampling event dated December 19, 2003.

- U.S. EPA Region 10, 2003 drinking water sampling data and related reports (Bates numbers: TC 012347 RD through TC 012689 RD).

- Records received by Hartig Rhodes Hoge & Lekisch in response to a Freedom of Information Act request to the Alaska Department of Environmental Conservation on Kivalina's drinking water (Bates numbers: TC 012155 RD through TC 12311 RD).

- Fish tissue data from the Alaskan Department of Fish and Game (ADF&G).

- O'Hara et al. 2003. Investigation of heavy metals in a large mortality event in caribou of Northern Alaska.

- Exponent report: *Evaluation of Metals Concentrations in Caribou Tissues*, December 2002 and poster presentation of this report by Garry et al. at the 2004 Society of Toxicology annual meeting.

- Alaska Division of Public Health study: *Public Health Evaluation of Exposure of Kivalina and Noatak Residents to Heavy Metals from Red Dog Mine* (ADPH 2001).

- Alaska Department of Environmental Conservation Alaska Water Quality Criteria Manual for Toxic and Other Deleterious Organic and Inorganic Substances. As amended through May 15, 2003. http://www.state.ak.us/dec/dawq/wqs/documents/aquaticlifecriteriacyanide.htm.

- Maps of rivers and sampling station locations.

- Declarations of Caleb Adams, Enoch Adams, Leroy Adams, Andrew Koenig, Jerry Norton, and Joe Swan in support of motion for partial summary judgment. Case No. A02-231, U.S. District Court for the District of Alaska at Anchorage.

- Depositions of Enoch Adams, Jr. (July 8, 2003); Jerry Norton, Leroy Adams, Andrew Koenig, and Joseph Swan, Sr. (July 9, 2003). Case No. A02-231, U.S. District Court for the District of Alaska at Anchorage.

- Declaration of Philip Driver. Case No. A02-231, U.S. District Court for the District of Alaska at Anchorage.

- U.S. EPA. 1998. Authorization to discharge under the National Pollutant Discharge Elimination System, Cominco Alaska, Inc. (Red Dog Mine). 29 July. U.S. Environmental Protection Agency, Region 10.

- Red Dog hydrograph data for 1999 through 2003.

- U.S. EPA. Undated. Findings of No Significant Impact (FONSI). Attached to EPA Public Notice: EPA Proposes Reissuance of Teck Cominco Alaska, Inc. (Red Dog Mine), Issuance Date: March 31, 2003. http://yosemite.epa.gov/R10/WATER.NSF/0/996828b449c2dd8e88256cfb0058236f?Open. Accessed November 11, 2004, last updated April 1, 2003. U.S. Environmental Protection Agency, Region 10.

- Teck Cominco Alaska Incorporated. 2003. Red Dog Mine NPDES Permit AK-003865-2 Reauthorization Request. February.

- Report by Alfred L. DeCicco, Alaska Department of Fish and Game. Life History of Anadromous Dolly Varden (*S. malma*) in Northwestern Alaska. Prepared for the 1990 meeting of the International Society of Arctic Char Fanatics in Murmansk, USSR. September 16–22, 1990.

- Thompson, M. 2002. Letter to Kathleen Collins, EPA, regarding the ionic composition of water. December 8.

- Sanitary Survey. Prepared for the Village of Kivalina and the Kivalina Subsistence Committee, by ASCG, Inc., May 2004.

- Declarations of Robert Moran, September 25, 2003, and November 16, 2003. Expert Statement of Robert Moran, December 2003. Case No. A02-231, U.S. District Court for the District of Alaska at Anchorage.
- Data on cyanide speciation in Station 001 water samples collected from May 30 to July 1, 2003.
- Thiocyanate concentrations from Outfall 001 and Station 10 in 1997 and from Outfall 001 in 2001.

9. I have also relied on my knowledge of the scientific and medical literature pertaining to the relationship between exposures to these constituents and the potential for adverse health effects.

IV. Basis for Opinions

10. My opinions are supported by my evaluation of the available site monitoring data based on accepted methodology for assessing risk to human populations (e.g., U.S. EPA 1989; Faustman and Omenn 2001) and the peer-reviewed scientific and toxicological literature. Comments regarding illness or disease observed by community members provided an insufficient basis for evaluating potential health effects related to the alleged discharge violations because of the non-specific description of health problems and lack of quantitative information such as individual exposures, rates of health problems, and controls for other causes.

A. Wulik River Water Samples

11. Discharge of water from the Red Dog Mine is associated with high-water-flow events during spring snow melt and summer rains. Water from the mine is discharged to the Middle Fork of Red Dog Creek, which flows into the main stem of Red Dog Creek, which flows into Ikalukrok Creek, which in turn flows into the Wulik River (summarized in the U.S. EPA [2003b] environmental assessment). Due to historically elevated levels of metals prior to the development of the Red Dog Mine, Ikalukrok Creek is not classified for drinking water use between its confluences with Red Dog Creek and the Wulik River. Therefore, the focus of evaluation for potential human health effects is data from sampling Stations 1 and 2 on the Wulik River. Drinking water for the Village of Kivalina is obtained at monitoring Station 1 on the Wulik River, about 30 miles downstream of Station 2. Station 2 is located just downstream of the confluence with Ikalukrok Creek. Data from 2002 to 2004 are also available from Station 3 of the Wulik River upstream of the confluence with the Ikalukrok River.

12. The constituents listed in the complaint (i.e., cadmium, TDS, cyanide) were named because of possible concentrations of these constituents in samples collected at the point of discharge that exceeded the limits specified in the discharge permit for the mine. Nevertheless, exceedance of a permit limit concentration does not equate to a human health risk in the receiving water or downstream. The permit limits for cadmium and cyanide are based on water concentrations that are protective of aquatic life, and the limit for TDS is based on not exceeding one-third of the natural background concentration in the receiving water. Levels of these constituents for protection of aquatic life and the

permit limits are much lower than levels that would result in health effects from water consumption by humans.

13. The permit limit for cyanide is based on the presence of the more toxic form of cyanide, "free" cyanide (e.g., HCN and CN^-). Alleged violations of the permit limits are associated with the measured concentration of "total" cyanide. A more relevant comparison to the permit limit, however, is free cyanide or weak-acid dissociable (WAD) cyanide (i.e., more soluble cyanide compounds that more readily dissociate to free cyanide in weak acid conditions). Concentrations of WAD cyanide in the receiving water from the mine were considerably less than the total cyanide concentration, which includes forms that are considerably less soluble or bioavailable, and less toxic. Conversion of iron cyanides to HCN by UV radiation is not an environmentally important source of free cyanide in flowing rivers, due to low penetration of natural UV light through surface waters and rapid volatilization of HCN (Wuerthele 1999).

14. Free cyanide is generally accepted in the toxicological literature to be the more toxic form of cyanide (ATSDR 1997, 2004a,b; Hartung 1994; Smith 1996; Wuerthele 1999). Acute, high-dose cyanide poisoning affects the ability of the cells in the body to use oxygen. At lower but sufficiently elevated doses for long-term exposure periods (chronic), the primary effects associated with cyanide are potential neurotoxicity and effects on the thyroid, but not cancer (Philbrick et al. 1979; NTP 1993; ATSDR 1997, 2004a,b). Humans and many other animals are equipped to deal with low levels of exposure to cyanide through well-known cyanide detoxification mechanisms (Blakley and Coop 1949; Way 1984; Smith 1996; Parkinson 2001). Free cyanide and other cyanide

compounds also occur naturally in the environment, as well as in some foods. For example, fruits such as peaches, apples, cherries, and apricots contain cyanide in their pits, and juices made from these fruits contain measurable levels of cyanide (reviewed by ATSDR 1997, 2004a,b). The natural presence of cyanide in foods, however, does not necessarily mean that eating these foods would cause health effects. Low doses of cyanide do not saturate the detoxification mechanisms that prevent cyanide toxicity, and therefore are not a health concern (U.S. EPA 1981).

15. Strong complexes of metal cyanides that are not included in the WAD cyanide measurement have low solubility, which likely reduces their bioavailability once ingested and therefore limits the amount of free cyanide that enters or is released into the body.

16. Thiocyanate, which is an interfering agent in the total cyanide analysis (ATSDR 2004a), are of low acute toxicity in mammals (Pettigrew and Fell 1973; Solomonson 1981; Hartung 1994; Smith 1996). Thiocyanate occurs naturally in many foods (ATSDR 1997; ATSDR 2004a); in fact, it is the form produced by mammals in the detoxification of cyanide and is a biomarker for cyanide exposure (Blakley and Coop 1949; Pettigrew and Fell 1973; Solomonson 1981; Way 1984; Parkinson 2001; ATSDR 2004a,b). Antidotes given to humans for cyanide poisoning increase the rate of detoxification by increasing the conversion of cyanide to thiocyanate, which is readily excreted through the urine.

17. Chronic, long-term oral exposure of rats to sodium thiocyanate by food did not produce evidence of carcinogenicity (Lijinsky and Kovatch 1989). Similarities in some of the endpoints for chronic toxicity of free cyanide and thiocyanate may be due to the

production of thiocyanate by cyanide detoxification. For example, administration of 2,240 ppm of thiocyanate or 1,500 ppm of potassium cyanide in the diet of rats for up to 11 months produced neurological and thyroid effects, although potassium cyanide (but not thiocyanate) was associated with reduced body weight gain (Philbrick et al. 1979), and chronic liver and kidney effects in rabbits were attributed to a cyanide dose that exceeded the detoxification capacity of the enzyme that converts cyanide to thiocyanate (Okolie and Osagie 1999). U.S. EPA Region 9 (U.S. EPA 2004d) lists a preliminary remediation goal for thiocyanate in tap water that is 2.5 times higher (less stringent) than the corresponding level for free cyanide.

18. Thus, the alleged permit violations do not constitute scientific evidence of human health risk. To assess whether human health risks may possibly be associated with discharges from the mine during the alleged violation period, a screening-level risk assessment was conducted of the Wulik River sampling data from 1998 to the present. Specifically, the concentrations of these constituents in water were compared to EPA drinking water standards (cadmium and cyanide) or other health-based information (TDS and sulfate, which lack health-based standards).

19. The initial health basis for setting EPA drinking-water standards (maximum contaminant limit or MCL) for chemicals such as cadmium and cyanide involves calculating a drinking-water equivalent level (DWEL), which is the concentration that one could drink in 2 liters of water per day, 365 days per year, for a lifetime and not exceed a chemical's EPA chronic "reference dose." The reference dose is the daily dose at and below which no health effects would be anticipated for up to a lifetime of exposure, even in sensitive

populations (U.S. EPA 2004b,c). Reference doses are typically set well below levels at which health effects might occur and account for scientific uncertainties by the use of uncertainty factors to increase the margin of "safety." EPA furthermore set more stringent MCLs for cadmium and cyanide than their lifetime health-protective concentrations (i.e., DWELs): 25 percent of the DWELs for cadmium and for cyanide (U.S. EPA 2004a).

20. The cyanide reference dose and MCL is additionally protective, because it is based on the "free" form of cyanide, even though the MCL is applied to measurements of total cyanide, which include more than the most toxic "free" form (U.S. EPA 1985, 2004c).

21. Thus, the comparison of the MCLs for cadmium and cyanide in drinking water to even the maximum river water concentration measured between 1998 and September 2004 represents a very conservative screening for potential risk, because one would not be able to drink 2 liters of water containing the maximum concentration every day for a lifetime. The average concentration at a sampling station is therefore a more realistic comparison to drinking-water criteria such as MCLs, which assume long-term exposure.

22. None of the monitoring data in the Wulik River from 1998 to September 2004 for cadmium, total cyanide, or WAD cyanide exceed federal primary drinking-water standards (MCLs) for municipal water supplies (Table 1 in Attachment B). Most of the measurements for cyanide were undetectable. Although total cyanide levels were not measured at Station 1 near Kivalina Village after 1994, cyanide levels were measured upstream at Station 2 at the confluence of the Ikalukrok Creek with the Wulik River.

Cyanide levels (resulting from discharge from the mine) at Station 1 are expected to be less than at Station 2, which is closer to the mine.

23. Although thiocyanate was not measured in the Wulik River, thiocyanate was measured at Outfall 001 at the mine outfall and Station 10 downstream of the mine in the main stem of Red Dog Creek before its confluence with the Ikalukarok Creek. The thiocyanate concentration at Station 10 in 1997 was 1.4 mg/L, which is lower than the EPA (2004d) preliminary remediation goal for thiocyanate in tap water for human consumption of 1.8 mg/L. More recent measurements of thiocyanate at Station 10 were not available; however, levels measured at Outfall 001 in September of 2001 indicated lower thiocyanate levels than were measured previously at this station in 1997. Thiocyanate levels resulting from mine discharge would be considerably lower downstream in the Wulik River. Therefore, no human health concern for thiocyanate in the Wulik River is indicated.

24. The TDS concentration includes all the dissolved forms of chemicals, including metals, minerals, and other naturally occurring constituents of water (e.g., calcium, magnesium, chlorides, sodium, potassium, carbonate, bicarbonate, sulfate). The majority of TDS is made of these latter constituents, which are of low toxicity to humans. Concentrations of metals and other constituents potentially from the mine that are of regulatory concern for health or environmental effects are also quantified separately. The primary components of TDS in discharge water from the mine are sulfate (72 percent) and calcium (24 percent) (Thompson 2002; U.S. EPA 2003b). However, downstream of the mine at

Stations 2 and 1, sulfate makes up only 28 percent and 23 percent of TDS on average, respectively (Thompson 2002).

25. TDS has a secondary standard of 500 ppm for drinking water and no primary standard. Secondary standards are based on aesthetics such as taste, odor, or staining and are not health-based. Consequently, secondary standards are actually non-enforceable guidelines and are not predictive of levels at which health effects might occur. Of the major constituents of TDS, none has primary drinking-water MCLs based on human health effects, and only sulfate has a secondary standard (250 ppm), which is based on odor and taste. Numerous studies in animals (including neonatal animal models for infants) and human volunteers, and epidemiological studies of populations with high sulfate levels in their drinking water have demonstrated that the health effects of sulfate are relatively mild, and the chief concern is its laxative effect in unacclimatized people and infants at water levels exceeding 1,000 ppm (summarized by U.S. EPA 2003a).

26. U.S. EPA (2003a) notes that a CDC panel favored a health advisory level of 500 ppm for exposure of unacclimatized people and infants to sulfate in drinking water. This level, however, appears to be considerably lower than necessary to prevent laxative effects in sensitive populations, because it was based on an older case study involving three infants who developed diarrhea when fed formula mixed with water containing sulfate at levels ≥630 ppm and extremely high TDS levels of 2,474 to 3,123 ppm (Chien et al. 1968; U.S. EPA 2003a); such levels are not present in the Wulik River. Because the laxative effect of water depends on the total amount of osmotically active constituents of TDS in water (e.g., substantial amounts of magnesium were present; Chien et al. 1968), sulfate was

likely not the only constituent involved. Based on the available scientific studies and reviews, the secondary standards for sulfate and TDS are lower than necessary to protect against the potential short-term laxative effects from these constituents in water (Gomez et al. 1995; Esteban et al. 1997; Henningsen 1997; Heizer et al. 1997; CDC and U.S. EPA 1999; U.S. EPA 2003a). None of the samples in the Wulik River water from 1998 to 2004 exceeded the secondary standards for sulfate or TDS (Table 1). These results indicate that sulfate and TDS levels in the Wulik River are well below levels that might be expected to cause even mild short-term laxative effects in sensitive people.

B. Kivalina Drinking Water

27. Drinking water for the Village of Kivalina was sampled by Teck Cominco in December 2002. The water in the drinking-water system was drawn from the Wulik River Station 1 in August 2002, during the time of year that the mine was discharging water. Water from the Wulik River is typically drawn in late summer and stored for use through the winter. Cadmium, total cyanide, and WAD cyanide were all undetectable at detection limits that were well below the standards described previously. Water samples were also collected in December of 2002 from Wulik River Station 1 and from the Kivalina River located 4 miles north of the Village of Kivalina. The mine was not discharging at this time. The Kivalina River does not receive flow from waters that receive Red Dog mine discharges. Concentrations of cadmium and cyanide (total and WAD) from these river samples likewise were undetectable. TDS levels were similar among the Kivalina drinking water and Wulik and Kivalina river water samples and were approximately half of the secondary MCL for TDS. Less than 25 percent of the TDS concentration was sulfate.

Therefore, sulfate levels were well below the secondary standard for odor and taste, and the higher advisory level proposed by EPA for potential concern for laxative effects of sulfate.

28. Sampling of Kivalina Village water by EPA in May 2003 and by ASCG in July 2003 found similar results for these constituents.

29. ADPH (2001) likewise concluded, based on earlier sampling of Kivalina drinking water and Wulik River water, that "[o]verall, the levels of heavy metals in the drinking water are very low and do not pose a health threat."

C. Fish

30. Tissue samples of Dolly Varden were collected and analyzed for metals by ADF&G in spring and fall of 1998 through 2003. The other named constituents, TDS and cyanide, were not analyzed by ADF&G, likely because these constituents are not a concern for accumulation in fish tissue or for food-chain risks to humans. TDS constituents are osmotic parameters that would be regulated in fish tissues, and cyanide does not accumulate in fish tissues because it is metabolized (U.S. EPA 1985; ATSDR 2004a,b). Cadmium in fish muscle tissue was undetectable in all samples. Cadmium was detected in fish liver and other organs (e.g., kidney, reproductive organs) that may be eaten by local tribal people. However, concentrations were relatively low and do not constitute a health concern. For example, based on the maximum concentration measured in any liver sample over this time period (2.12 ppm dry weight; 0.44 wet weight), and assuming that

fish liver is about 2.3 percent of body weight (ADPH 2001), one would have to eat nearly four fish livers containing this maximum concentration every day for nearly a lifetime to reach and exceed the EPA reference dose for cadmium in food (U.S. EPA 2004b). As noted above, the reference dose is the dose at and below which there is no concern for health effects, even in sensitive populations. This scenario is unrealistic, however, because based on the sampling data, one would not be able to eat more than four fish livers per day at this maximum concentration for years. This concentration was the highest measured in 72 fish sampled in 6 years. The next highest sample concentration was about half the maximum concentration. At the average concentration of cadmium in fish liver measured during this time period (0.13 ppm wet weight), one would have to eat 13 fish livers every day for nearly a lifetime. Calculations for other organs yield similar conclusions, that the amount of tissue consumption needed to reach the reference dose level would be improbable. Therefore, the fish tissue data for the Wulik River do not indicate a human health concern from cadmium.

31. ADPH (2001) concluded, based on ADF&G fish tissue data from 1990 through 2000, that "metals are not bioconcentrated up the food chain" and that "concentrations of heavy metals measured in fish collected from streams and rivers in the Red Dog Mine area are well below any risk-based screening level and do not pose a public health concern."

D. Caribou

32. Caribou are one of the primary subsistence food sources for native northern Alaskans. Caribou range widely and may occasionally drink water from the rivers that receive flow

from the mine. However, the available tissue data from caribou collected in the mine area have not indicated increased levels of cadmium or other metals in caribou.

33. ADPH (2001) reviewed tissue data for metals from caribou collected near the Red Dog Mine in 1996 (O'Hara et al. 2003) and concluded, "The average concentrations of metals found in caribou harvested near the Red Dog Mine and haul road were low. Eating caribou from the Western Arctic Caribou Herd does not pose a public health threat." Cadmium levels in muscle, liver, or kidney of caribou from Red Dog Mine were lower than or similar to levels of caribou from other northern Alaska locations (O'Hara et al. 2003).

34. Teck Cominco, in collaboration with the ADF&G, collected additional caribou tissue samples near Red Dog Mine operations and along the DeLong Mountain Regional Transportation System (haul road) in April 2002. The tissue samples of the 10 caribou collected in 2002 showed no significant elevations in tissue metals (including cadmium) concentrations relative to previous samples from the area, and no consistent elevations compared to caribou elsewhere in northern Alaska, or in caribou and reindeer from Canada and Scandinavia (Garry et al. 2004).

E. Summary

35. The available site data show no evidence of a human health concern as a result of the permit violations for discharges at the mine alleged in the complaint.

V. Exhibits Supporting Opinions

An exhibit supporting or summarizing my opinions is attached as Attachment B. The exhibit is as follows:

- Table 1. Summary of Wulik River 1998–2004 monitoring data (Stations 1 and 2) for cadmium, cyanide, TDS, and sulfate.

I may change the format of the information depicted in the above exhibit, use additional information drawn from the materials considered in forming my opinions, and/or use additional exhibits at trial.

VI. Compensation

36. Dr. Tsuji is employed at Exponent, Inc. The company is being compensated at the rate of $290.00 per hour of Dr. Tsuji's time spent preparing her opinions.

VII. Other Testimony

37. Dr. Tsuji's testimony within the preceding four years includes the following:

Name of Case	Court	Trial or Deposition Testimony
United States v. ASARCO INCORPORATED et al. Coeur d'Alene Tribe v. ASARCO INCORPORATED et al.	U.S. District Court for the District of Idaho	Deposition
Keith Herd, et al. v. ASARCO INCORPORATED, et al.	U.S. District Court for the Northern District of Oklahoma	Deposition; Proceedings
David Douglas and Karen Douglas v. Unocal Corporation, et al.	Eighth Judicial District Court, State of New Mexico, County of Taos	Deposition

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